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Prognosis in patients with diphtheric myocarditis and bradyarrhythmias: assessment of results of ventricular pacing

Benjamin A Stockins, Fernando T Lanas, Juan G Saavedra, Juan A Opazo

Abstract

***Objective*—To determine the prognosis in patients with diphtherial myocarditis and bradyarrhythmias and to assess the results of ventricular pacing in those with third degree atrioventricular block.**

***Design*—Case series.**

***Setting*—Referral department of cardiology in a teaching hospital.**

***Patients*—Twenty four out of 46 patients admitted with diphtherial myocarditis over 10 years had bradyarrhythmias. Six had sinus bradycardia, 15 atrioventricular or intraventricular conduction disturbances, and three atrioventricular dissociation.**

***Main outcome measure*—Death rate.**

***Results*—Eleven patients died (46%): all seven patients with third degree atrioventricular block, the patient with bifascicular block, and three of the six patients with bundle branch block. Seven died of cardiogenic shock and four of ventricular fibrillation. All nine patients with sinus bradycardia or atrioventricular dissociation survived.**

***Conclusion*—Conduction system disturbances in patients with diphtherial myocarditis are markers of severe myocardial damage and a poor prognosis. In addition, ventricular pacing does not improve survival.**

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The incidence of diphtheria has decreased in most countries. The disease persists, however, in some rural areas of southern Chile,[1] as well as in other developing countries,[2–4] and has recently been described in homeless alcoholics in Seattle, Washington.[5]

Myocarditis induced by the exotoxin remains one of the most dangerous complications of this disease. It develops in 10–25% of patients and has a mortality as high as 60%.[6] The most common signs of diphtherial myocarditis are electrocardiographic changes such as ST depression and ST inversion, disturbances in atrioventricular and intraventricular conduction, and signs of heart failure. The diphtherial exotoxin has a high affinity for the conduction system of the heart.

Conduction disturbances have been associated with a high mortality, but ventricular pacing has not been adequately evaluated in patients with third degree atrioventricular block. We analysed the prognosis of patients with diphtherial myocarditis and bradyarrhythmias and assessed the results of ventricular pacing in those who developed third degree atrioventricular block.

Patients and methods

Diphtheria was diagnosed in 167 patients in the 10 years from 1976 to 1986 at the University Hospital in Temuco (capital of the ninth region of Chile). Diagnosis was based on clinical findings of a membrane in the pharynx and positive results on culture of a throat swab. Forty six patients were admitted to hospital with diphtherial myocarditis.

Myocarditis was diagnosed on the basis of clinical signs of heart failure, electrocardiographic changes, or arrhythmias. In this study we analysed the course of disease in 24 patients who developed one of four features: sinus bradycardia of less than 50 beats per minute, disturbances in atrioventricular or intraventricular conduction, and atrioventricular dissociation.

The patients had a mean age of 7 years (range 4–15 years); 14 were girls and 10 boys. No patient had been immunised against diphtheria.

All patients were taken to the intensive care unit and received specific treatment for diphtheria as well as haemodynamic support for cardiac failure. All patients with high degree atrioventricular block (bifascicular block or third degree atrioventricular block) received a ventricular pacemaker.

Results

Of the 167 patients with diphtheria, 46 developed diphtherial myocarditis (27%). Twenty four of the patients with myocarditis developed bradyarrhythmias (52%). Six patients developed sinus bradycardia, three atrioventricular dissociation (atrioventricular junctional rhythm with a higher rate than the sinus node), one left anterior hemiblock, four right bundle branch block, two left bundle branch block, one bifascicular block (left anterior hemiblock and right bundle branch block), and seven third degree atrioventricular block.

Eight of these patients developed other arrhythmias at some stage in their disease. Premature ventricular beats were the most common, developing in six patients. One

Department of Cardiology, School of Medicine, Universidad de la Frontera, Temuco, Chile
B A Stockins
F T Lanas
J G Saavedra
J A Opazo

Correspondence to:
Dr B Stockins, Facultad de Medicina Universidad de la Frontera, Casilla 54-D, Temuco, Chile.

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patient developed atrial fibrillation and another paroxysmal supraventricular tachycardia.

Eleven of the 24 patients with bradyarrhythmias (46% of the group) died: all seven patients with third degree atrioventricular block, the patient with bifascicular block, and three of the six patients with bundle branch block. The patient with left anterior hemiblock survived.

All the patients with sinus bradycardia or atrioventricular dissociation survived. The prognosis in those with right bundle branch block was the same as those with left bundle branch block: half of the patients in each group died.

Despite electrical pacing, all patients with advanced atrioventricular block died. Three of them died less than 12 hours after pacing started, two between 12 and 24 hours, and three between 24 and 48 hours. The cause of death was cardiogenic shock in seven and ventricular fibrillation in four.

Discussion

The three main findings of our study were (*a*) that patients with advanced atrioventricular block died, and ventricular pacing did not improve survival; (*b*) that patients with bundle branch block had a high mortality (50%); and (*c*) that sinus bradycardia and atrioventricular dissociation had a good prognosis.

Our findings in patients with third degree atrioventricular block are similar to those in other studies,[7 8] but ventricular pacing was not assessed in these studies. Unfortunately, pacing did not improve the prognosis in these patients. To our knowledge, only one study has reported the survival of a patient with third degree atrioventricular block after ventricular pacing; he survived with a left anterior hemiblock.[9]

The dismal prognosis in patients with third degree block can be explained by the severe deterioration of systolic function which accompanies atrioventricular block. Therefore, despite electrical stimulation of the ventricle, the mechanical response is insufficient, most patients dying of cardiogenic shock. This is similar to the high mortality in patients with anterior myocardial infarction and high grade conduction disturbances. Since patients with diphtheria can theoretically recover as the inflammation wanes, however, evaluation of other temporary haemodynamic support manoeuvres such as aortic counterpulsation seems reasonable. The high mortality from bundle branch block can be explained similarly, with the block representing significant deterioration in ventricular function during systole.

The mechanism of sinus bradycardia in children with diphtheria is not clear. Possible explanations include increased vagal tone (in which case bradycardia would not represent an inflammatory phenomenon) or the effect of the exotoxin on the right atrium and sinus node.

The good prognosis observed in patients with atrioventricular dissociation is probably related to the different mechanism of this arrhythmia. This condition does not represent a block in atrioventricular conduction but rather acceleration of a secondary pacemaker in the ventricle to a rate faster than sinus rhythm. The cause of this arrhythmia in diphtheria is also not clear, but it is probably due to an increase in automaticity in the ventricle or His bundle that occurs in inflamed myocardium.

We conclude, as have other workers,[6 7] that bundle branch block and complete heart block in patients with diphtherial myocarditis are markers of severe myocardial damage and a poor prognosis. In addition, cardiac pacing is unlikely to improve survival.

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